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THE DIFFERENTIATION OF CARDIAC AND PULMONARY DYSPNEA

Dyspnea may be defined as the feeling of labored breathing or shortness of breath. It is thus a manifestation of ventilatory insufficiency even though the etiology may be neurogenic, metabolic, cardiovascular or pulmonary.

When advanced pulmonary disease exists with minimal cardiac disease or when advanced cardiac disease exists with minimal pulmonary disease, the differentiation between pulmonary and cardiac etiology of dyspnea can be made on the basis of the general clinical evaluation. When both diseases coexist, however, as frequently happens in the older age group, the clinical evaluation of each with respect to its contribution to the symptom of dyspnea may be difficult if not impossible. Extensive tests of pulmonary and cardiocirculatory function provide a more comprehensive understanding of these complicated conditions. In this paper various ideas and tests will be discussed, which, in the Cardio-Pulmonary Laboratory at Bellevue Hospital, have been found useful in differentiating cardiac and pulmonary factors in patients who are dyspneic at rest or after mild exercise.

The physiologic measurement which shows the best correlation with the feeling of dyspnea is the ratio of the breathing reserve to the maximal breathing capacity. The maximal breathing capacity is the maximal amount of air which the subject can take in and give out of his lungs per unit of time, and the breathing reserve is the maximal breathing capacity minus the actual ventilation in any given state. Dyspnea, regardless of etiology, involves a decrease in maximal breathing capacity or an increase in actual ventilation or both. Patients with pulmonary disease ordinarily complain of dyspnea when the breathing reserve is less than 65 or 70 per cent of the maximal breathing capacity. In patients with cardiac disease the relationship is less constant, and dyspnea sometimes persists when the breathing reserve is more than 70 per cent of the maximal breathing capacity.

The maximal breathing capacity is determined by having the subject breathe in and out as fast as possible at a depth resulting in the largest possible volume of ventilation per unit of time, as measured on a recording spirometer. When performed properly, the test is strenuous, and in order to avoid fatigue and acapnia it is usually carried out for only twelve to twenty seconds. Normal values for males range around 150 liters per minute and for females around 100 liters per minute. The ability to move air in and out of the lungs per unit of time is dependent on the integrity of the thoracic cage, including bones, joints, and muscles; on the size and elasticity of the lungs and pleura; on the patency of the airways from the mouth and nose to the diffusing surface of the lungs and on neuromuscular stimulation and coordination. The volume of the lungs constitutes the spacial boundaries which limit ventilation while the other factors are more directly concerned with the rate at which ventilation can proceed within these boundaries. In chronic pulmonary disease the reduction in maximal breathing capacity is usually related to pulmonary fibrosis or emphysema or both. In cardiac disease the reduction is due in part to pulmonary vascular congestion and edema, causing impairment of expansibility and retractibility of the lungs. Contrary to the finding in primary pulmonary disease, patients with pulmonary vascular congestion due to cardiac disease probably have a decrease in maximal breathing capacity during exercise because of an increase in the degree of congestion. The muscles involved in ventilation also may become fatigued during exercise because of inadequate circulation. For these reasons the actual value of the maximal breathing capacity during exercise may be lower than the previously determined value, causing the breathing reserve after exercise to appear higher than it is. This explanation may help to account for the finding that dyspnea sometimes persists in patients with cardiac disease when the breathing reserve as calculated is more than 70 per cent of the maximal breathing capacity.

The actual ventilation, which is the minute volume of ventilation in any given state of rest or exercise, may conveniently be measured at rest, during one minute of exercise, and during each of five minutes of the recovery period. The actual ventilation is the resultant effect of the various respiratory stimuli, including acid-base relationships, anoxia, proprioceptive stimuli from the lungs, heart and great vessels, stimuli from other parts of the body and emotional stimuli. The actual ventilation is the total ventilation as distinct from the alveolar ventilation, which is that part of the total ventilation which takes part in gas exchange. The total ventilation minus the alveolar ventilation leaves the amount of ventilation which flushes in and out of the physiologic dead space. In patients with pulmonary disease the actual or total ventilation is often increased because the dead space ventilation takes up a larger than normal proportion of the total. In patients with pulmonary vascular congestion the actual ventilation also is increased, possibly as a result of afferent nervous impulses from the left atrium, pulmonary veins, pulmonary capillaries or adjacent pulmonary tissue. Congestion of these structures may initiate afferent impulses, possibly mediated by the same fibers as the Hering-Breuer reflex, giving rise to an increase in ventilation.

Since pulmonary vascular congestion has been implicated both in reducing the maximal breathing capacity and in increasing ventilation, it is obvious that pulmonary vascular pressure relationships are intimately related to the question of dyspnea. These relationships differ in chronic pulmonary disease, in right ventricular failure, and in left ventricular failure. Interstitial fibrosis of the lungs, for example, may surround and constrict the smaller pulmonary vessels and reduce the cross section of the pulmonary vascular bed to such an extent that an increase in pulmonary arterial pressure is required to maintain flow. In emphysema, increased vascular resistance may result in part from a reduced capillary bed due to obliteration of alveolar walls. Right ventricular failure secondary to pulmonary disease

is associated with an increase in right ventricular diastolic and right atrial pressure in addition to right ventricular systolic and pulmonary arterial pressure. Clinically this situation corresponds to cor pulmonale with failure of the right side of the heart. Failure of the left ventricle involves elevation of left ventricular diastolic pressure, and in order to maintain flow from right to left there must also be elevation of left atrial, pulmonary venous, pulmonary capillary, pulmonary arterial, and right ventricular systolic pressures. Knowledge of both pulmonary venous and pulmonary arterial pressures would be extremely helpful in distinguishing between pulmonary disease and left ventricular failure, but unfortunately pulmonary venous pressure cannot ordinarily be measured in man. Knowing pulmonary arterial pressure is not enough since it may be elevated in either pulmonary or cardiac disease.

Probably elevation of pulmonary venous and capillary pressure is regularly present when dyspnea results from cardiac disease and is not present when dyspnea results from pulmonary disease. Using this criterion, one would not expect right ventricular failure to cause dyspnea, and indeed it is probable that right ventricular failure alone does not cause dyspnea of significant degree, although of course dyspnea due to associated pulmonary disease is frequently seen in patients with cor pulmonale and right ventricular failure. If congenital abnormalities are excepted, the cardiac diseases causing dyspnea may for practical purposes be narrowed down to left ventricular failure, mitral stenosis and insufficiency, and other mechanical processes interfering with the entrance of blood into the left side of the heart in patients whose right ventricles are still strong enough to build up an elevated pressure in the pulmonary circuit. The necessity for resorting to indirect evidence in the evaluation of pulmonary venous and capillary pressures is obvious since these vessels are ordinarily out of reach of the exploring venous catheter. Let us discuss some of the indirect evidence which helps in evaluating the etiology of dyspnea.

Measurement of the total capacity of the lungs and its subdivisions helps to determine the type of pulmonary pathology. For example, in pulmonary emphysema the residual air, which is the volume of air remaining in the lungs at the end of a maximal expiration, ordinarily exceeds 35 per cent of the total capacity of the lungs. Both residual air and total capacity are usually greater than normal in absolute terms. Pulmonary congestion and edema due to left ventricular failure, on the other hand, are usually associated with low values for residual air and total capacity because much of the air space is taken up by the congested vascular bed. Differentiation on the basis of pulmonary volumes is inconclusive since there are also forms of pulmonary fibrosis with normal or low residual air volumes, in which dyspnea occurs because of loss of pulmonary elasticity. Furthermore, both pulmonary vascular congestion and emphysema may exist at the same time.

The alveolar ventilation, or a figure related to it, provides information regarding the activity of the respiratory center in the brain. The partial pressure of carbon dioxide (pCO_2) in the arterial blood provides an accurate measure of the partial pressure of carbon dioxide in the alveolar air, and this in turn varies inversely with the alveolar ventilation. When alveolar ventilation is inadequate, the pCO_2 in arterial blood exceeds the normal value of 40 mm. of mercury and when the alveoli are overventilated, arterial pCO_2 is less than 40 mm. In pulmonary emphysema the arterial pCO_2 tends to be elevated, and in a general way the amount of elevation is related to the degree of emphysema. The highest values for pCO_2 are seen in patients in whom cor pulmonale and right heart failure are associated with severe emphysema. Congestion of the lungs due to cardiac disease in the absence of primary pulmonary disease is more likely to be associated with a low pCO_2. The latter finding is sometimes noted in rheumatic heart disease with lesions of the mitral valve and may be related to afferent impulses to the respiratory center initiated by elevated pressures in the left side of the heart, the pulmonary veins, pulmonary capillaries or edematous pulmonary parenchyma. Pulmonary embolism and infarction, even of small extent, may likewise be associated with increased alveolar ventilation and low arterial pCO_2. A low pCO_2 is thus suggestive of vascular congestion of the pulmonary veins and capillaries or of pulmonary embolism, while a high pCO_2 is suggestive of pulmonary emphysema with or without right ventricular failure in addition. With any figure related to ventilation, it is obvious that metabolic, neurologic, and emotional factors must be taken into consideration.

The oxygen saturation of the arterial blood is related to the arterial pCO_2 since alveolar ventilation is the most important single factor in maintaining adequate oxygenation of the arterial blood. Accordingly, the conditions associated with a low arterial pCO_2 are in general associated with a high oxygen saturation of the arterial blood. This applies to mitral valvular disease or left ventricular failure of moderate severity. In frank pulmonary edema the presence of fluid in the alveolar walls and smaller airways may lead to varying degrees of unsaturation of the arterial blood. Pulmonary disease, particularly of the emphysematous type, frequently causes lowering of the oxygen saturation of arterial blood as a result of poor correlation between alveolar ventilation and alveolar perfusion with blood. In certain exceptional cases in which diffusion of oxygen from alveolar air to capillary blood is markedly impaired, the oxygen saturation of arterial blood may be low in spite of a normal pCO_2.

Recent studies for which the direct Fick technic was employed have demonstrated that the cardiac output does not increase significantly during exercise in cases of cardiac decompensation. This finding indicates that the failing heart is incapable of pumping more blood than that which is required in the resting state. In pulmonary disease exercise tolerance may be greatly impaired, but an increase in oxygen consumption calls forth an appropriate increase in cardiac output, as judged by comparison with the normal. The response of the cardiac output to exercise may thus help in establishing the diagnosis of cardiac decompensation in the presence of pulmonary disease.

The determination of the intake of oxygen during a standard exercise test has proved to be helpful in evaluating cardiac function in patients with a confusing mixture of cardiac and pulmonary factors. The test is performed by having the patient step onto and down from a 20 cm. stool thirty times in one minute and by collecting the expired air during the exercise. Normally the intake of oxygen is found to be at least 480 ml. per minute per square meter of body surface. In patients with severe pulmonary disease in whom the oxygen saturation of the arterial blood falls after the step test there may be a moderate lowering of the oxygen intake. Experience has shown, however, that in patients with a significant cardiac factor the intake of oxygen falls to 350 ml. or less per square meter. The accepted physiologic explanation is as follows: The one minute step exercise produces an unsteady state during which oxygen intake through the lungs does not keep pace with the energy expenditure of the tissues. Under these conditions the blood flow through the lungs is the major factor determining the amount of oxygen taken up in the lungs. Accordingly the finding of a low intake of oxygen indicates subnormal blood flow and implies impaired cardiac function. It is only on the basis of experience that one can decide at what point individual variation in cardiac response ends and impairment of cardiac function begins. Such experience strongly suggests that an oxygen intake of less than 350 ml. per square meter during the specific test described is due to inadequate functional capacity of the heart.

The possibility of deriving a figure related to blood

flow from a simple exercise test is of special interest because the determination of cardiac output by the venous catheterization technic is a relatively elaborate and time-consuming affair while the step test with analysis of the expired air could readily be done as an office procedure. The conclusions arrived at from the step test are consistent with those derived from direct Fick determinations during exercise in that both tests indicate that the cardiac output is inadequate during exercise in patients with cardiac decompensation.

Therapeutic tests may be used to a limited extent as an aid in differential diagnosis. The response of the patient with cardiac failure to digitalization sets him apart from the patient with pulmonary disease uncomplicated by a cardiac factor. Other types of therapy, such as administration of antibiotics, bronchodilators or diuretics and positive pressure breathing, may be of help in selected cases.

Analysis of the physiologic factors involved in cardiac and pulmonary disease provides a background on the basis of which clinical signs and symptoms can be more critically appraised. For example, the observation that patients with pulmonary emphysema often breathe most easily in the supine position while patients with cardiac dyspnea are relieved by sitting upright can be interpreted along the following lines. In the flat position the weight of the abdominal contents exerts more pressure from below on the diaphragm and in this way more complete expiration is facilitated. In emphysema the diaphragm tends to be low and flat, and there is usually bronchiolar narrowing which causes special difficulty in expiration. It is, therefore, physiologically understandable why emphysematous patients might breathe more easily in the supine position. The fact that an abdominal binder, by adding pressure on the diaphragm from below, may give further relief lends credence to the explanation. In the supine position the volume of blood in the lungs is considerably larger than in the upright position, however. This finding is related to the fact that blood from below the heart is more readily returned to the right ventricle when the antagonistic effect of gravity is eliminated. An increase in the volume of blood in the lungs is associated with increased pulmonary vascular congestion and is disadvantageous to the patient with cardiac dyspnea. This patient accordingly chooses a more upright position.

Since the physiologic abnormalities in cardiac disease are in many respects different from those of pulmonary disease, it is to be expected that the subjective responses to the diseases would be different. Some of these differences tend to be overlooked because of the similarity of the predominant symptom which is often shortness of breath. In pulmonary disease shortness of breath frequently occurs in relatively pure form and may rightly be termed "dyspnea." In cardiac disease, however, shortness of breath is usually associated with a feeling of extreme weakness, probably related to tissue anoxia due to inadequate blood flow. This may be localized in the muscles which have been used or within the chest, or it may be an overall sensation throughout the entire body. There also may be a feeling of suffocation in which the patient has the urge to breathe but feels unable to do so. Tachycardia may provide another source of discomfort to the patient with cardiac disease. Such symptoms cannot rightly be included under the term "dyspnea," although true dyspnea is often an associated phenomenon. The information gained from a careful symptomatic history may be useful in differential diagnosis but must be tempered by an awareness of the vagaries of subjective responses. The emotional reaction of the patient to the disease is such an important and variable factor that the correlation of symptoms to physiologic findings sometimes may be impossible.

In conclusion, it must be emphasized again that dyspnea is a manifestation of ventilatory insufficiency which may be caused by certain types of pulmonary or cardiac disease. A reasonable estimate of the contributions of each disease to the dyspnea can be made on the basis of characteristic physiologic abnormalities.

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